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"PERIARTHRITIS" OF THE SHOULDER AND CORONARY DISEASE*

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FOR SOME TIME we have been interested, clinically, in the painful stiff shoulder. Of particular interest is the large group where the etiology is uncertain, and the onset of shoulder pain often insidious. The condition may progress through an acutely painful phase leading to varying degrees of stiffness or "frozen" shoulder. Examination reveals, as well as variable limitation of movement, tenderness along the tendon of the long head of the biceps and over the lateral aspect of the shoulder. The x-ray is usually negative or may show a diffuse osteoporosis of shoulder girdle bone.

In general, these cases do not come to necropsy for histological study and many of the present concepts have been garnered from gross appearances at open operation. These visual impressions are imperfectly supported by pathological data. The multitude of names given the condition reflect the confusion in clinical minds. They include terms such as Duplay's disease, frozen shoulder, scapulo-humeral arthritis, neuritis, adhesive capsulitis, periarticular fibrositis, myositis, peritendinitis, periarthritis and others.

Numerous authors[1 to 5] have observed clinically the association of shoulder disability with severe coronary disease. Ernstene and Kinell[2] found painful shoulder in 17 or 12% of 138 cases of myocardial infarction. The severity of the condition ranged from typical periarthritis to mild, aching pain. The left shoulder was affected more often than the right. Symptoms persisted for weeks or months. Schott[3] discussed six cases of coronary disease with painful disability of one or both shoulders, and concluded the condition resembled scapulo-humeral arthritis. Fishberg[4] suggested coronary disease be kept in mind in all middle-aged individuals who complain of pain and limitation in the shoulder, especially the left. Johnson[5] found the shoulders, hands or both were affected in 39 of 178 consecutive cases of myocardial infarction and called the condition "post-infarction sclerodactylia".

In our series of 35 routine autopsies in whom the subacromial bursa and biceps tendon sheath were examined, 6 cases of severe coronary disease were included. Four of the coronary cases showed lesions in the subacromial bursa and 2 of these in the biceps sheath as well. The only other individual showing significant lesions suffered from chronic rheumatoid arthritis with bursal involvement.

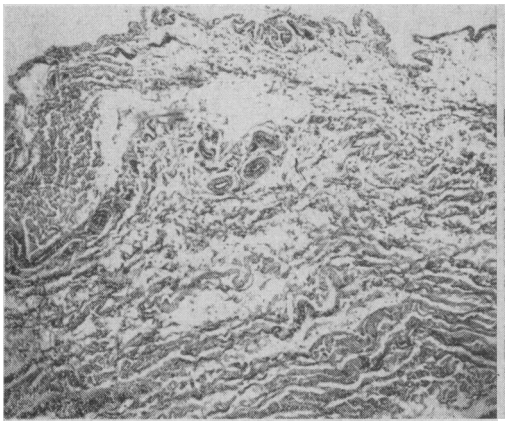

Fig. 1

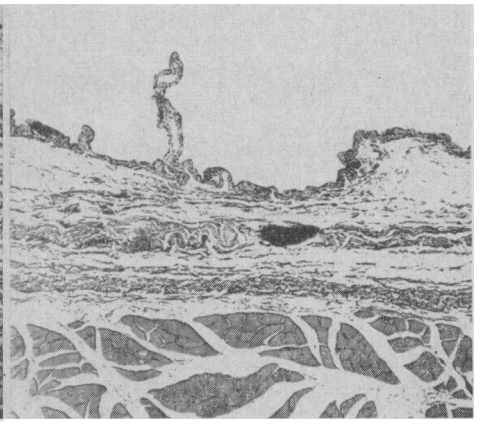

Fig. 2

Fig. 1.—Subacromial bursa, x 34. Normal: average thickness and density.
Fig. 2.—Subacromial bursa, x 34. Chronic invalid.

A short discussion of the normal histology will help to introduce the pathology found:

Histology.—The normal subacromial bursa, and bursal extension along the biceps tendon, is lined by cells, often a single layer thick but, in some areas, two or three or more cells thick. The nuclei are flattened, elongated, or polygonal in shape, and do not always form a continuous layer. The lining is often thrown into small folds, or delicate fronds. Deep to this layer is a loosely packed, collagenous fibrous tissue layer, with

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variable intervening fatty and areolar layers running parallel to the surface. Small arterioles are arranged in groups, and lie in the collagen tissue just deep to the lining layers. The walls of these arterioles normally are thicker than comparable arterioles elsewhere. The bursal walls vary considerably in thickness. In general, it was found that muscular active individuals had thicker bursæ. Chronic invalids, or elderly patients, had a thinner, more areolar type wall. Fig. 1 demonstrates a wall of average thickness and Fig. 2 the bursa of a chronic invalid, showing the fine, areolar type structure with a delicate lining.

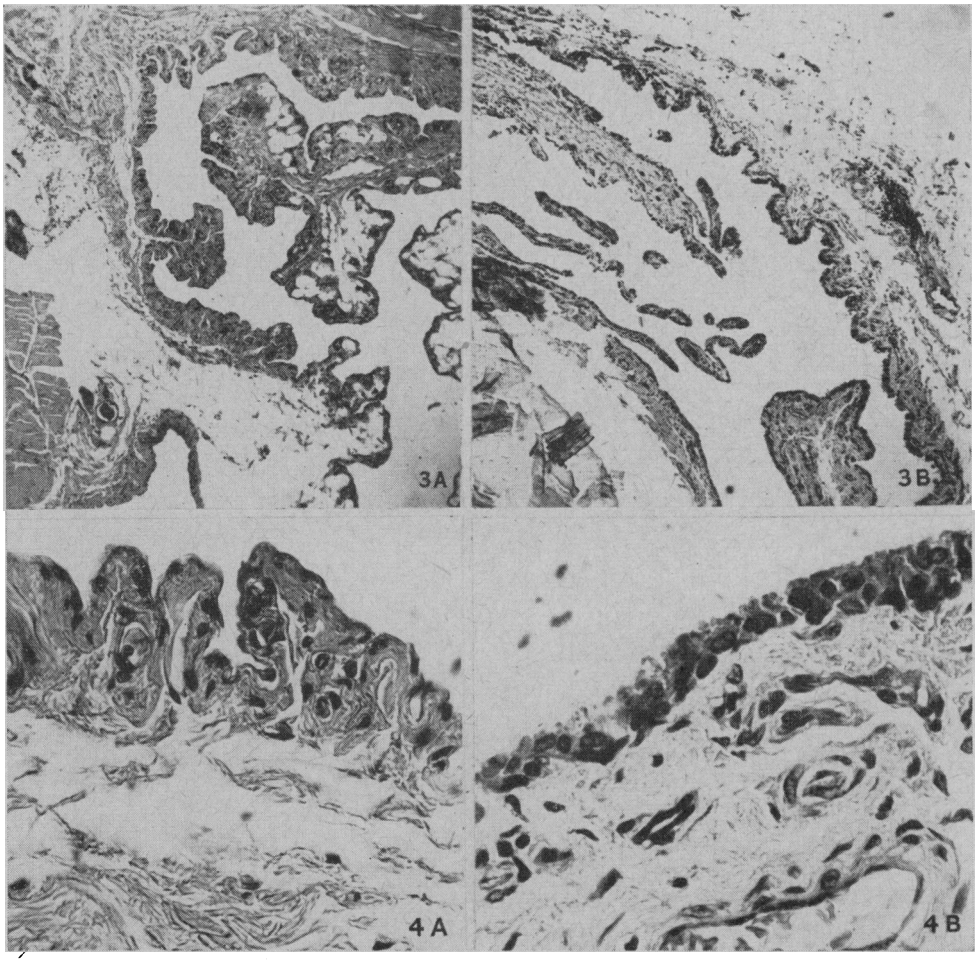

Fig. 3.—Bursal sheath long head of biceps tendon, x 34. (A) Normal individual. (B) Case 1—note the regimentation of the lining cell layer. A perivascular collection of small round cells is present. **Fig. 4.**—Bursal sheath long head of biceps tendon, x 344. (A) Normal synovial lining cells. (B) Case 1—lining cells are intensely active and arranged in a columnar like fashion. Vessels are engorged.

We will now describe the alterations found in the cases of coronary disease:

CASE 1

H.N. was a male, age 57, with severe luetic heart disease and aortic insufficiency. He suffered anginal attacks for 1½ years before death, with pain referred to both shoulders. He gradually developed increasing stiffness and pain in the shoulders, beginning about 8 months before death which, despite sympathetic blocks, did not improve. Eventually, examination revealed marked limitation of scapulo-humeral movement, together with acute tenderness over the biceps tendon in its groove. X-rays of the shoulder showed no abnormality. Symptoms were improving with physiotherapy, when he died suddenly. At autopsy, no infarction was present. The heart weighed 1,150 grams. There was considerable coronary atherosclerosis, without complete occlusion, and luetic intimal thickening about the coronary ostia. This was considered sufficient to cause coronary insufficiency. The supraspinatus tendon appeared normal. Gross examination of the bursa revealed thickening of the walls, which were bunched up in folds, with intervening areas of normal thickness. No adhesions were demonstrated from floor to roof, The sheath of the long head of the biceps tendon was opaque and thickened as well; however, a synovial cavity could be demonstrated.

Histology of subacromial bursa.—The bursal wall is two to three times the normal thickness in some areas, with fairly normal wall intervening. In the thickened areas the surface layer of the synovia has disappeared, and it, as well as subjacent layers, is replaced by firm, cellular, vascular fibrous connective tissue, broad bands of which run in an oblique fashion with intervening fatty tissue layers. Vessels of arteriole size are thin-walled and engorged, with loss of normal grouped arrangement. Occasional groups of perivascular lymphocytes are present.

Bursa of long head of biceps tendon.—In serial sections, two phases of inflammation are seen side by side. In some areas there is intense activity of the lining cells which are regimented and increased in number, forming an almost columnar-type of lining. Thin-walled blood vessels are engorged, and focal collections of perivascular lymphocytes are present (Figs. 3B and 4B). The appearance is that of active inflammation. In other areas there is gross thickening, due to replacement of the synovial lining and subsynovial tissues by a cellular fibrous connective tissue. In this fibrous connective tissue are areas of hyaline degeneration.

Deltoid muscle.—Scattered single muscle fibres are atrophic. Around these individual fibres there is proliferation of perimysial cells.

appeared fairly normal. No adhesions from floor to roof were demonstrated. The long head of the biceps tendon sheath was opaque and thickened, and adhesions could be seen firmly binding the bursal walls together.

Histology—subacromial bursa.—Areas of marked thickening of the wall alternate with a wall of fairly normal thickness (Fig. 5). In the thickened regions there is often loss of synovial lining layer, and the entire wall consists of dense vascularized cellular fibrous connective tissue. In this, multiple focal areas of hyaline degeneration are present. Fibrosed villi, which have been transected, also show areas of hyaline degeneration. The occasional perivascular collection of lymphocytes is seen. In some areas a new synovial lining appeats to have redifferentiated over the fibrosed surface.

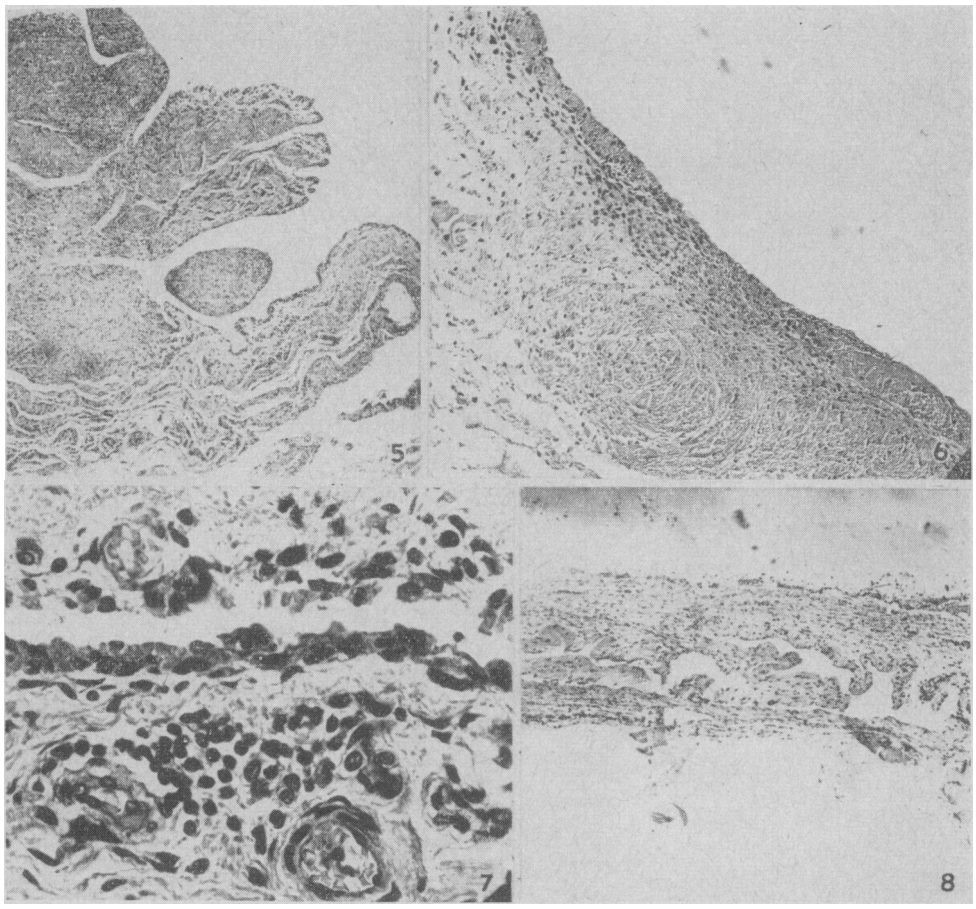

Fig. 5. (Case 2).—Subacromial bursa, x 34. The wall is grossly thickened and fibrosed. Areas of hyaline degeneration are present. New synovial lining has redifferentiated over some areas. **Fig. 6.** (Case 2).—Subacromial bursal wall, x 80. A limited area of acute necrosis of lining cells. **Fig. 7.** (Case 2).—Bursa long head of biceps tendon, x 343. Intense activity of lining cells is apparent. A focal collection of perivascular lymphocytes is present. **Fig. 8.** (Case 2).—Bursa long head of biceps tendon, x 34. (Central tendon has fallen out in preparation). Both layers of bursa are fibrosed and numerous fibrous adhesions bind them firmly together.

CASE 2

Male, age 61. In the six months before death, this patient suffered two severe myocardial infarctions, and was extremely restricted in his activities by angina, with substernal pain which radiated to both shoulders, and down both arms. He complained of stiffness and discomfort in the right shoulder, of some weeks' duration, though the time of onset is uncertain. On examination, there was pain on movement of the right shoulder, with marked limitation of external rotation. He died suddenly. At autopsy, old myocardial infarctions were present. The supraspinatus tendon appeared normal. The bursa appeared thick and opaque. The synovial wall was gathered up in thick folds, while the intervening wall

Over one limited area there is seen complete loss of differentiation and necrosis of the surface cells of the synovia (Fig. 6). This appears to represent an acute lesion.

Bursa of long head of biceps tendon.—Again, two distinct phases of inflammation are seen in adjacent areas. In some areas, (Fig. 8) there is intense activity of synovial lining cells which are arranged in an almost columnar fashion (as seen in Case 1). Small blood vessels are dilated and engorged, and scattered perivascular collections of lymphocytes are seen. The appearance is that of active inflammation. In adjacent areas, (Fig. 9) both layers of the bursal wall consist of dense fibrous tissue in which areas of hyaline degeneration are present.

There are multiple, firm fibrous adhesions binding the layers together. This would appear to be the chronic phase of an inflammatory process.

Deltoid muscle.—The occasional muscle cell is atrophic with local proliferation of perimysial cells. The lesion is minimal.

The remaining two cases show comparatively limited areas of fibrosis and hyalinization in the subacromial bursa of a similar nature to those described.

DISCUSSION

In 1872, Duplay[6] ascribed the pain and stiffness in periarthritis to an adhesive inflammation of the subacromial bursa. Since then, a good deal of attention has been paid to lesions in the musculotendinous cuff, apparently resulting in bursal inflammation and loss of gliding space.[7]

More recently, an increasing number of observers[8 to 12] have emphasized lesions in the bursal sheath of the long head of the biceps tendon, with lessened emphasis on the subacromial bursa. Lippmann,[8] at open operation, invariably found inflammatory appearances in the biceps tendon sheath in both acute and chronic phases of 32 cases of periarthritis. By contrast, he found adhesions in the subacromial bursa in only 2 of the 32 cases. Surgical release of the biceps tendon immediately released all shoulder restriction, and he considered no true contracture of joint capsule was present. Others[13, 14] have hypothesized the presence of cicatricial lesions in the capsule, or periarticular musculature.

Our cases represent, pathologically, what appears to be an inflammatory process in the biceps bursa and/or the subacromial bursa. There were no significant lesions in the overlying muscle. The minor changes that were present could well be ascribed to disuse atrophy.

The early stages are characterized by marked inflammatory activity of synovial cells in some areas, and necrosis of these cells and the adjacent collagen in others. This is presumably followed by the laying down of vascularized fibrous tissue, and may go on to adhesion formation. With subsidence of the acute inflammatory aspects, the bursa is left with abnormally thick and densely fibrotic areas over which a new synovium differentiates. These changes strikingly resemble those found in the synovium in chronic rheumatoid arthritis. Indeed, the bursa from the case of rheumatoid arthritis is almost indistinguishable from that of Case 2.

It seems reasonable that if shoulder movement is limited by bursal thickenings as well as pain, possibly arising from the rubbing together of inflamed and roughened areas in the sensitive synovial lining, that subsidence of the acute inflammation and reconstitution of synovium may, in part, explain the recovery that can occur. Persistence of pain may suggest continued areas of active inflammation as demonstrated in these cases.

SUMMARY

1. Histological examination of the subacromial bursa and synovial sheath of the long head of the biceps tendon was made in 35 routine autopsies.

2. No abnormality was found in 30 cases.

3. Six cases of severe coronary insufficiency were included in the series, four of whom showed pathological alterations in the biceps sheath and, or, the subacromial bursa. The remaining two cases had no history of shoulder disability.

4. The only remaining case showing bursal abnormality was a case of chronic rheumatoid arthritis.

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There is still considerable prejudice and many misconceptions both among physicians and laymen alike related to epilepsy. Great efforts are being made in public enlightenment, but it is fundamentally the duty and the responsibility of the physician treating the patient to assist social workers and other interested individuals and agencies in the rehabilitation of the patient and the adequate handling of his sociological difficulties.—I. S. Zfass, *Va. Med. Monthly*, 79: 188, 1952.